"A STUDY ON THE PREVALENCE OF PERIPHERAL VASCULAR DISEASE IN DIABETIC FOOT ULCER PATIENTS"

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

With partial fulfilment of the Regulations

For the Award of the Degree of

M.S.(General Surgery),

Branch -I

GOVERNMENT KILPAUK MEDICAL COLLEGE

CHENNAI-600 010

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled **" A STUDY ON THE PREVALENCE OF PERIPHERAL VASCULAR DISEASE IN DIABETIC FOOT ULCER PATIENTS"** is a bonafide and genuine research work carried out by me under the guidance of Prof.Dr.R.Vasuki M.S., Department of General surgery, Kilpauk Medical College, Chennai-10.

This dissertation is submitted to **THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI** in partial fulfilment of the degree of M.S. General Surgery examination to be held in May 2018.

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CERTIFICATE BY THE GUIDE

This is to certify that the dissertation titled **" A STUDY ON THE PREVALENCE OF PERIPHERAL VASCULAR DISEASE IN DIABETIC FOOT ULCER PATIENTS"** is a bonafide research work done by **Dr. SATHEESH KUMAR M** , postgraduate in M.S. General Surgery , Kilpauk Medical college, Chennai -10 under my guidance and supervision in my satisfaction, in partial fulfilment of the requirements for the degree of M.S. General Surgery.

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INSTITUTIONAL ETHICS COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE,
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Protocol ID. No.06/2016 Meeting held on 14/12/2016
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval **"A STUDY ON THE PREVALENCE OF PERIPHERAL VASCULAR DISEASE IN DIABETIC FOOT ULCER PATIENTS"** submitted by Dr.Satheesh Kumar.M., Post Graduate in MS (General Surgery), Govt. Kilpauk Medical College, Chennai.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

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INTRODUCTION

Peripheral Vascular Disease is a chronic limb ischemia caused by atherosclerosis of the peripheral arteries. In diabetic patients, atherosclerosis occurs prematurely and progress at an accelerated pace commonly involving crural arteries namely tibials and peroneals with baring of the arteries of the foot. The perception of muscle pain in the lower limbs on exercise which is the most common symptom of Peripheral Vascular Disease may be blunted in diabetic patients by the presence of peripheral neuropathy. Therefore a patient with diabetes and Peripheral Vascular Disease is more likely to present with an ischemic ulcer or gangrene than a patient without diabetes.

The characteristic vascular involvement in diabetes had made it possible to carry out vascular reconstruction where proximal vessel like popliteal is anastomosed to foot vessels like

dorsalis pedis thus bypassing the obstructed tibial and peroneal vessels. This pedal artery bypass techniques has led to a significant decline in the incidence of all levels of limb amputations.

HISTORICAL BACKGROUND

- Jose Goyanes (1876-1964) of Madrid used vein grafts to restore arterial flow.
- In 1908, Friedrich Trendelenburg (1844-1924) attempted Pulmonary embolectomy.
- Barney Brooks (1884-1952) , Professor of Surgery at Vanderbilt University in Nashville, Tennessee, initially introduced clinical angiography and femoral arteriography in 1924.
- Reynaldo dos Santos (1880-1970), a Portuguese urologist, reported the first translumbar aortogram 5 years later.
- Rene Leriche (1879-1955) proposed an arteriectomy for arterial thrombosis in 1937 and later, periarterial sympathectomy to improve arterial flow.
- Leriche also described a syndrome of aorto-iliac occlusive disease in 1940.

AIM OF THE STUDY

- To evaluate the occurrence of Peripheral Vascular Disease in diabetic foot ulcer patients.
- Ankle-Brachial Index (ABI) which is calculated by dividing the ankle systolic pressure by the brachial systolic pressure provide a measure of blood flow to the ankle. Ankle-Brachial Index measurement in diabetic patients attending Surgery Outpatient Department for foot ulcer and associated complaints could help early detection of Peripheral Vascular Disease thus making the initiation of early therapy and reducing the risk of critical limb ischemia and limb loss possible.

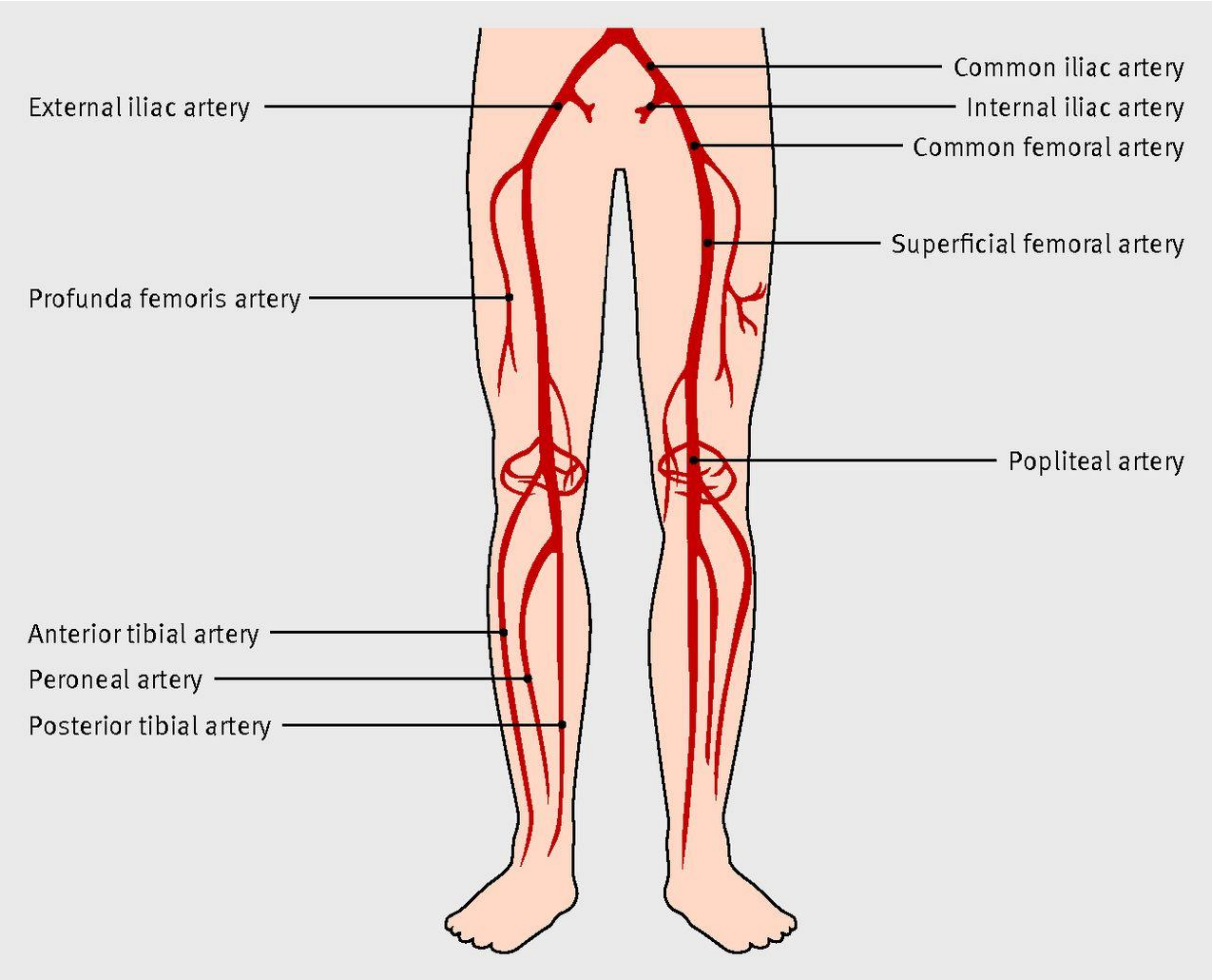
PERIPHERAL VASCULAR DISEASE

Peripheral Vascular Disease (PVD) commonly referred to as Peripheral arterial disease or Peripheral artery occlusive disease refers to the obstruction or deterioration of arteries other than those supplying the heart and within the brain. The common denominator among a number of pathological processes that manifest their effects on the arterial circulation is the impairment of circulation and resultant ischemia to the end organ involved.

Significant disability and loss of function from PVD result in an enormous cost in impaired quality of life for the aging population and a direct financial cost to the health care system.

Risk of PVD is increased in smokers and in patients with Hypertension, dyslipidemia, hypercoagulable states, renal insufficiency and diabetes mellitus. The risk of PVD also increases in individuals older than 50 years, male, obese or with a family history of vascular disease, heart attack or stroke.

ARTERIES OF LOWER LIMB



ARTERIES OF LOWER LIMB

Abdominal aorta bifurcates at the level of fourth lumbar vertebra (corresponds to the level of the umbilicus in anterior abdominal wall) into two common iliac arteries.Common iliac artery is about 5 cm in length; passes downward and laterally; and at the level of lumbo-sacral intervertebral disc, anterior to sacro-iliac joint, it divides into external and internal iliac arteries. Internal iliac artery supplies pelvic organs.

External iliac artery continues as common femoral artery at the level of inguinal ligament.About 5 cm below the inguinal ligament common femoral artery divides into superficial femoral and deep femoral (Profunda femoris) artery.Deep femoral artery provides collateral circulation around the knee joint and also communicates above with gluteal vessels to maintain collateral circulation around the gluteal region.

Superficial femoral artery at the hiatus in the adductor magnus, continues as popliteal artery up to the inferior angle of the popliteal fossa where it divides into anterior and posterior tibial arteries.

Anterior tibial artery supplies anterior compartment of leg and ankle, continues as dorsalis pedis artery which forms dorsal arterial arch of the foot. Posterior tibial artery supplies posterior compartment of leg and ends as medial and lateral plantar arteries which forms plantar arterial arch of the foot. Posterior tibial artery gives peroneal artery which runs close to fibula supplying calf muscles.

VASCULAR WALL ANATOMY

The arterial wall consists of three concentric layers:

1. The innermost layer is the intima. This is structurally a tube of endothelial cells in which the long axis of each cell is oriented longitudinally. The cells are aligned in a single

layer and interface with the blood, providing metabolic reactivity and signalling via transport of mediators through their internal cellular architecture. The intima is separated from the media by the internal elastic membrane.

2. The media is the major structural support for the artery. It is composed predominantly of circumferentially arranged smooth muscles ,collagen, elastin and proteoglycans. Proteoglycans are formed of disaccharides bound to protein; they serve as binding or cement material in the interstitial spaces. The blood supply for the inner part of the media is by direct diffusion through the intima whereas the outer part is supplied by smaller penetrating arteries known as vasa vasorum. The media is separated from the outermost layer, the adventitia, by the external elastic membrane.
3. The adventitia contains fibroblasts, collagen and elastic tissue and is the strength layer of the artery.

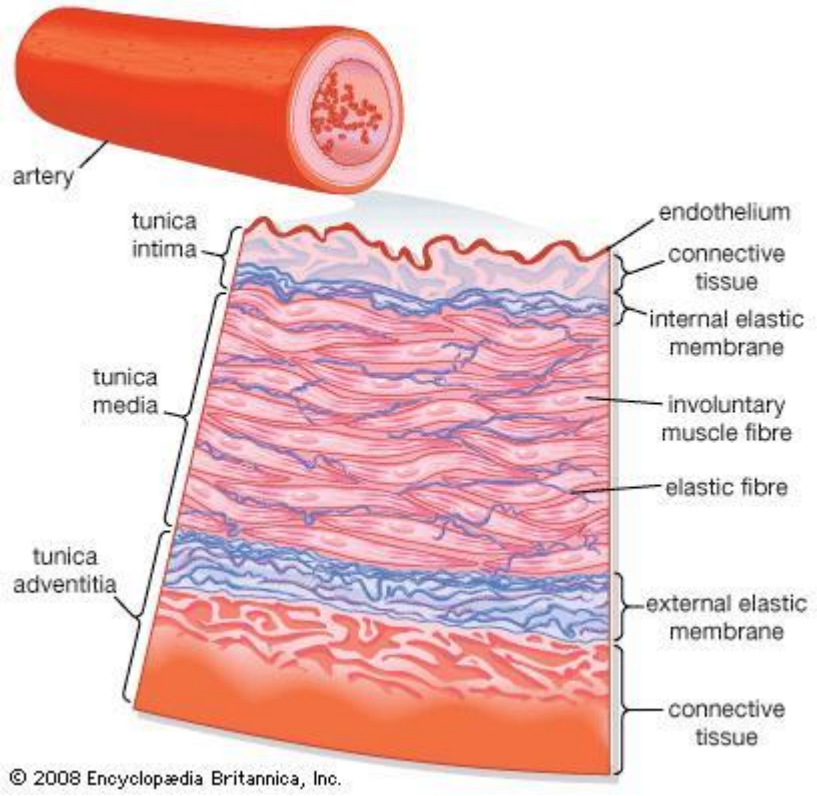
PATHOPHYSIOLOGY OF PVD

Atherosclerosis is the most common pathology associated with PVD.

Atherosclerosis is a hardening of an artery specifically caused by an atheromatous plaque. Hyperlipidemia, Hypercholesterolemia, Hypertension, Diabetes mellitus and exposure to infectious agents or toxins such as from cigarette smoking are all important and independent risk factors. The common mechanism is thought to be endothelial cell injury, smooth muscle cell proliferation, inflammatory reactivity and plaque deposition.

There are several components found in atherosclerotic plaque-lipids, smooth muscle cells, connective tissue and inflammatory cells often macrophages. Lipid accumulation is central to the process and distinguishes atheromas from other arteriopathies.

ARTERIAL ANATOMY

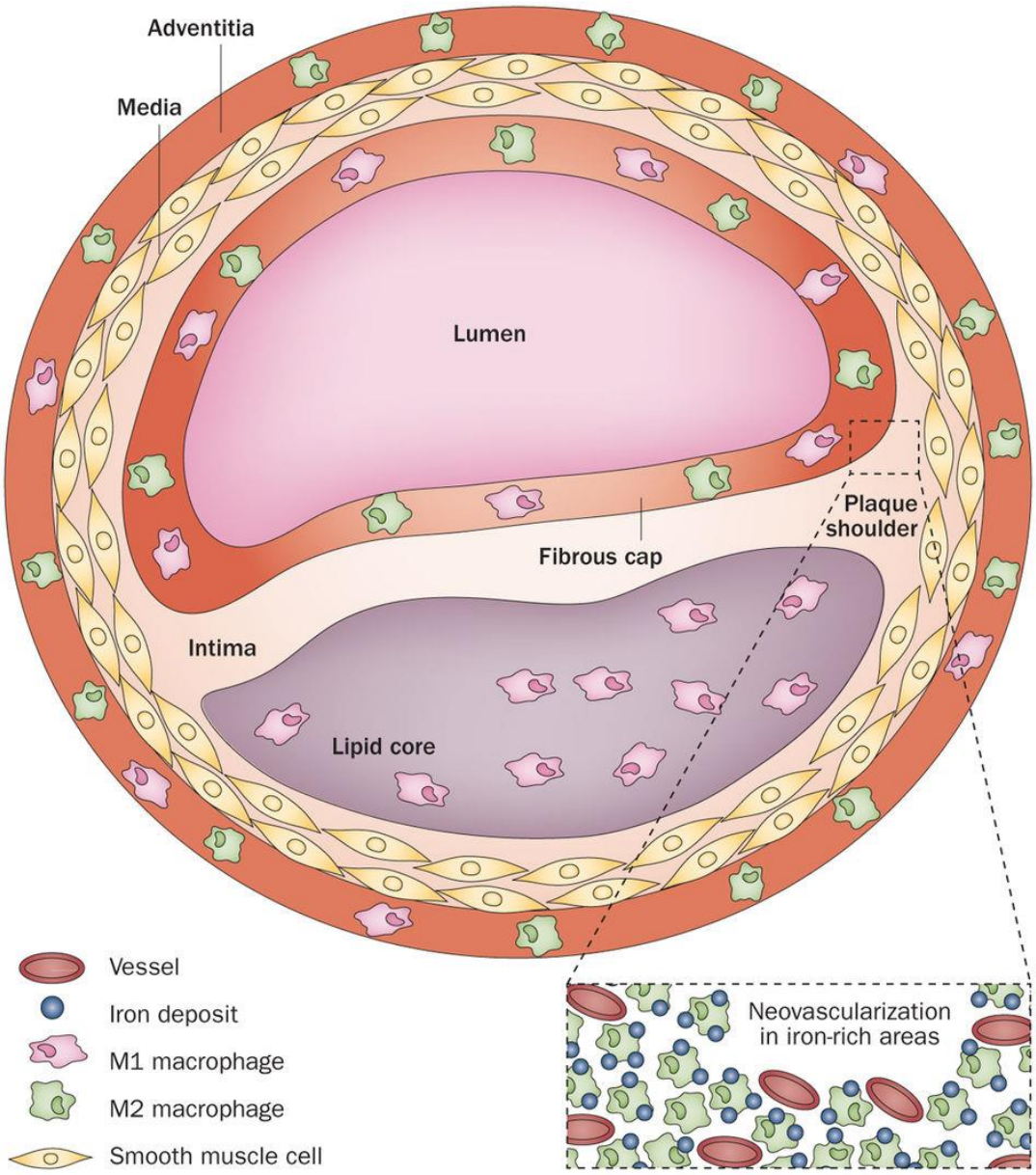


The plaque's lipid core may become a necrotic mix of amorphous extracellular lipids, proteins and prothrombotic factors covered by a layer of smooth muscle cells and connective tissue of variable thickness, the fibrous cap.

If the thin fibrous cap ruptures and the contents of the lipid core are exposed to circulating humoral factors, the body, perceiving the ulceration as an injury, may lay down platelets and initiate clot formation.

Ischemia can result from a number of possible plaque behaviours such as encroachment on the lumen (stenosis or narrowing) with hypoperfusion, stagnation and thrombosis; rupture of the fibrous cap inducing thrombus formation in the lumen, with outright occlusion; and embolization of thrombotic debris into the downstream circulation.

ATHEROSCLEROSIS



CHRONIC ARTERIAL INSUFFICIENCY

The clinical presentation ranges from asymptomatic to gangrenous tissue loss. Intermittent claudication is a common presentation in the outpatient setting and usually signifies mild to moderate vascular occlusive disease. Classically pain occurs with activity or ambulation and is relieved with rest. Because of the frequency of superficial femoral arterial disease, the usual location of the pain is in the calf, but claudication may also involve the thighs or the buttocks because the arterial disease may be located in the aorto-iliac segment. The arterial disease is usually one level above the symptomatic muscle group.

Patients who are limited in ambulation because of arthritis, severe lung disease or heart failure or who are diabetic with neuropathy may not experience leg pain and may present initially with advanced disease.Worsening

perfusion leads to critical limb ischemia (CLI), which may be manifested by rest pain. This is described as pain that occurs at rest; it may wake the patient from sleep. Pain occurs usually in the dorsum of the foot, relieved with dangling the leg over the edge of the bed. Patient may also have tissue loss with ulceration or non-healing wounds of the foot.

Detailed medical history of co-morbid conditions in addition to coronary artery disease (CAD), carotid artery stenosis (CAS), and prior stroke, risk factors for atherosclerosis (e.g., diabetes, hypertension, dyslipidemia, tobacco abuse, hyperhomocysteinemia) should be queried and their level of optimization understood.

ACUTE ARTERIAL OCCLUSION

Sudden occlusion of an artery is usually caused by an embolus. It may also happen when thrombosis occurs on an atherosclerotic plaque, although the outcome is usually

less dramatic because collaterals are likely to have developed in chronic arterial stenosis. Embolic arterial occlusion is an emergency that requires immediate treatment. Ischemia beyond 6 hours is usually irreversible and result in limb loss. The leg is often affected with pain, pallor, paralysis, loss of pulsation and paraesthesia. The limb is cold and the toes cannot be moved which contrasts with venous occlusion when muscle function is not affected. The diagnosis can be made clinically in a patient who has no history of claudication and has a source of emboli, who suddenly develops severe pain or numbness of the limb, which becomes cold and mottled. Movement becomes progressively more difficult and sensation is lost. Pulses are absent distally, but the femoral pulse may be palpable, even thrusting, as distal occlusion results in forceful expansion of the artery with each pressure wave despite the lack of flow.

CLINICAL CLASSIFICATION OF PVD

Boyd's classification of claudication:

Grade I	Patient complains of pain after walking. If the patient continues to walk, the pain subsides.
Grade II	Pain still persists on continuing walk; but can walk with effort.
Grade III	Patient has to take rest to relieve the pain.

Fontaine Classification:

STAGE	**CLINICAL**
I	Asymptomatic
IIa	Mild claudication
IIb	Moderate to severe claudication
III	Ischemic rest pain
IV	Ulceration or gangrene

Rutherford Classification:

GRADE	CLINICAL
0	Asymptomatic
1	Mild claudication
2	Moderate claudication
3	Severe claudication
4	Ischemic rest pain
5	Minor tissue loss
6	Major tissue loss

PHYSICAL EXAMINATION OF PVD

INSPECTION:

1. Change in colour is the most noticeable feature of an ischemic limb. Congestion and purple-blue cyanosed appearance particularly in dependency is noticed in chronic arterial insufficiency. The colours are quite

different from chronic congested extremity with venous insufficiency. When the limb is elevated it becomes pallor.

2. Signs of ischemia- Characteristic changes due to arterial insufficiency often occur at the most distal distribution of the concerned artery. These are thinning of the skin, diminished growth of hair, loss of subcutaneous fat, shininess, trophic changes in the nails which become brittle and show transverse ridges and minor ulceration on the pressure areas e.g. tips of toes, ball of the foot, heel and malleoli. It must be remembered that ulceration from venous insufficiency is virtually unknown below the level of the malleolus. To the contrary most ulcers from arterial insufficiency begin over the toes or at the most distal parts of the arterial trees. Ischemic ulcers are never seen on the leg or about the ankle without involvement of the toes.
3. Buerger's test- The legs of normal individual even if they be raised by 90 ^{o} remain pink. But in case of an ischemic limb elevation to a certain degree will cause marked pallor.

The angle between the limb at which pallor appears and the horizontal plane is known as Buerger's angle or the vascular angle. If the vascular angle is less than 30^{o} it indicates severe arterial occlusion.

4. Capillary filling time- After elevating the legs, the patient is asked to sit up and hang his leg down by the side of the table. A normal leg will remain pink. But an ischemic leg will change its colour from pallor to pink. This change of colour takes place slowly and is called the capillary filling time. An ischemic limb further changes its colour and becomes purple-red. This is due to the filling of dilated skin capillaries with blood.
5. Venous refilling- The extremities are elevated until collapse of vein has occurred. The extremities are then quickly lowered and the time required for the veins to refill particularly on the dorsum of the foot or hand is noted. Normally venous refilling will occur within 10 to 15 seconds. Longer time for filling means arterial

insufficiency. Time more than 1 minute denotes severe degree of arterial occlusion. This test cannot be performed in presence of venous varicosities with incompetent valves.

6. Pre-gangrenous state- The combination of rest pain, colour changes, hyperaesthesia with or without ischemic ulceration is frequently referred to as pre-gangrenous state.
7. Established gangrene- When the arterial occlusion is severe and is existing for quite a long time, gangrenous changes will be seen. This is the final stage and it is the dry gangrene which is characteristic of chronic arterial occlusion. It starts peripherally in the toes and extends proximally to involve gradually the entire foot and leg. Line of demarcation is often seen between the gangrenous part and the normal living limb. This line of demarcation is a line of inflammatory zone.

DIABETIC FOOT ULCERS

PALPATION:

1. Skin temperature of the ischemic limb is always colder than the normal limb.
2. Capillary refilling- The tip of the nail or pulp of a toe is pressed for a few seconds and the pressure is then released. The time taken for the blanched area to turn pink is a crude indication of capillary blood flow.
3. Venous refilling- The two index fingers are placed side by side on a vein. The fingers are now pressed firmly and the finger nearer to the heart is moved proximally keeping the steady pressure on the vein so as to empty a short length of vein between the two fingers. The distal finger is now released. This will allow venous refilling to be observed. This is poor in case of ischemic limb. This is known as Harvey's sign.
4. Palpation of peripheral pulses is the most important feature of the examination. In the lower extremity, the

femoral, popliteal, posterior tibial and dorsalis pedis pulses are felt.

5. The disappearing pulse- When peripheral pulses are apparently normal, exercising the patient to a point of claudication may unmask the effect of arterial obstruction and the previously palpable pulses may disappear. After rest of couple of minutes the pulse reappears. This is known as disappearing pulse. The reason is that exercise causes vasodilatation below the slight arterial occlusion. This needs more arterial flow to the distal part, so that the arterial flow which was already reduced cannot keep pace with the increasing demand, so that the arterial pressure falls and the pulse disappears.

AUSCULTATION:

1. Arterial bruits- A systolic bruit over an artery revealed by auscultation indicates slight occlusion of the artery. Such systolic bruit is conducted distally.

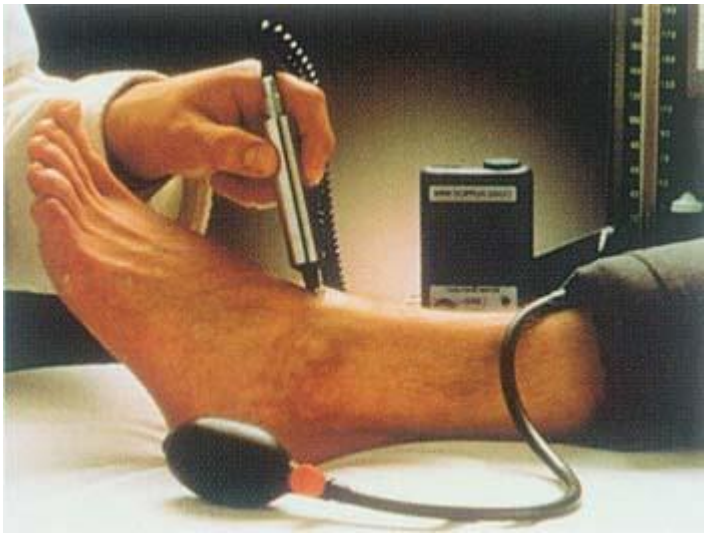
2. An oscillometer may be of some value in case of extremities with edema where peripheral pulses are difficult to palpate. This instrument consists of a blood pressure cuff attached to a manometer. This cuff is inflated to just above diastolic pressure. This evaluates pulsatile oscillations of the artery during systolic pressure. It has an advantage that it can quantify the degree of occlusion at bedside or even in the Out patient setting.

PHYSIOLOGIC TESTS FOR PVD

Tests commonly performed in the laboratory include the Ankle- Brachial Index (ABI) with multisegmental pressures & waveforms , Toe-Brachial index (TBI), Pulse Volume Recording (PVR), PhotoPlethysmography(PPG) and Arterial Duplex examination.

Regardless of plans for intervention, it is recommended that asymptomatic patients at risk for PVD and those with symptoms undergo ABI testing. This examination can be performed simply with a manual blood pressure cuff at the ankle and a continuous wave Doppler probe. With the patient in a supine position, after several minutes of rest to allow limb pressure to return to baseline, the cuff is inflated at the ankle, with the Doppler probe held at the location of the distal DP or PT signal. The systolic pressure is recorded as the pressure in the cuff when the Doppler signal returns. This process can be performed with multiple cuffs allowing for segmental pressure determination, which is helpful in localizing the level of the obstructing lesion. The ABI for a limb is calculated using the higher of the two ankle pressures divided by the higher of the two brachial pressures. Patients with an ABI of 0.90 or less have increased risk of PVD. Continuous wave Doppler analog waveforms can be obtained along with the segmental pressures.

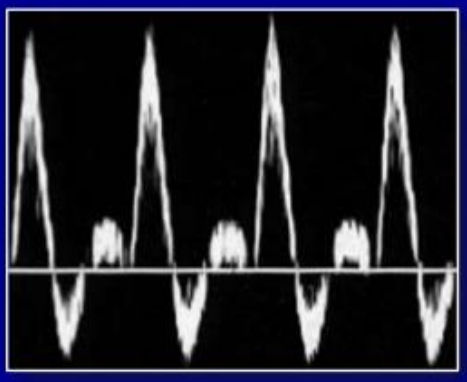
ABI MEASUREMENT



PPG uses an infra-red light emitting source and a photosensor; it is based on the principle that red light is decreased with increased blood flow in tissues to generate a pressure and waveform within the digit. The data generated from these studies should include bilateral brachial artery, high thigh, low thigh, calf, DP,PT and toe pressures with waveforms. A decrease in pressure of 20 to 30 mm Hg between adjacent segments is indicative of a significant lesion. The normal Doppler arterial waveform demonstrates triphasic flow with a sharp systolic upstroke, reversal of flow in early diastole from vessel compliance, and a low – amplitude forward flow throughout diastole. With obstructive disease the initial feature lost is the reversal of the flow component, leading to multiphasic (previously called biphasic) flow. Severe disease leads to blunting of the arterial waveform with decreased amplitude and decreased slope of the upstroke. With worsening symptoms, there is increased diastolic flow, resulting in monophasic flow. A change in

Normal triphasic waveform of peripheral arteries

Arterial high resistance flow



- Narrow frequency band
- Steep systolic increase
- Quick drop
- Early diastolic reverse flow (⅕ of systolic flow amplitude)
- Late diastolic short forward flow

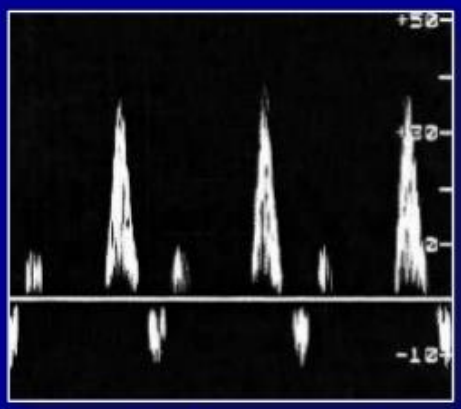
ABPI: Ankle Brachial Pressure Index

Stiegler H & Brandl R. Ultraschall in Med 2009 ; 30 : 334 – 363.

Hyperemic monophasic flow

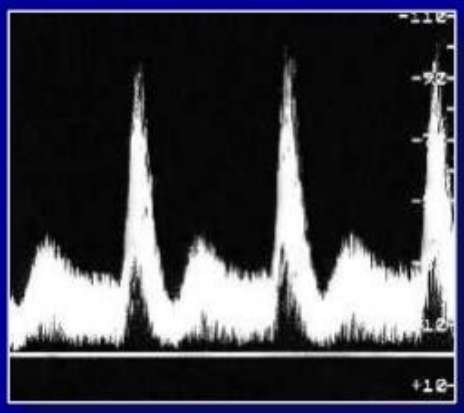
Following exercise

Normal DPA at rest



Normal triphasic waveform

Following exercise



Monophasic hyperemic flow

waveform can be interpreted, along with a change in pressure, as indicative of disease at that level.

Limitations of ABI and segmental pressure determinations include mural calcification, such as seen in DM and ESRD, leading to elevated pressures that do not accurately reflect intra-arterial perfusion pressure. With a non-compressible vessel, a TBI higher than 0.70 with an absolute digit pressure higher than 50 mm Hg , with a normal waveform , is indicative of preserved flow because digit arteries are relatively resistant to the intramural calcification. Symptomatic patients with palpable distal pulses or a normal resting ABI should undergo exercise testing with measurement of the post exercise ABI. The decrease in peripheral vascular resistance that occurs with exercise induced vasodilation will increase the drop in pressure seen across a stenotic lesion. Patients undergo resting ABI testing, followed by treadmill exercise until symptoms occur; repeat ABI testing may then reveal a decrease in ankle pressure of

20 mm Hg or a decrease in the ABI of 0.20. These changes , or a failure of the ABI to return to pre exercise baseline within 3 minutes, are interpreted as a positive result.

IMAGING STUDIES FOR PVD

When intervention is planned, further imaging to delineate the location and nature of disease is needed. Imaging studies include:

1. **Angiography:** This is the gold standard imaging study. Access is usually via the contralateral common femoral or left brachial artery. A complete diagnostic study is performed in four steps-
    a. Abdominal aortography, with a multiside hole catheter placed at the level of the diaphragm, imaging the abdominal aorta, celiac artery, superior mesenteric artery, inferior mesenteric artery and aortic bifurcation.

PORTABLE HAND HELD DOPPLER

b. Pelvic angiography with a multiside hole catheter at the aortic bifurcation, imaging the bilateral common iliac, hypogastric, and external iliac arteries, common femoral arteries and proximal superficial femoral (SFA) and profunda femoris artery.

c. The contralateral common femoral artery is then selected using an end- hole catheter and images of the contralateral SFA, profunda, popliteal, tibial, and pedal vessels are obtained in one three low bolus runs.

d. The access sheath is then pulled back to the level of the distal ipsilateral external iliac artery to image the ipsilateral limb.

Trans-stenotic pressure gradients and multiplanar images can clarify the significance of an ambiguous lesion. Complete assessment of the aortic and iliac inflow and bilateral lower extremities requires 75 to 100 ml of contrast.

2. **Computerised Tomography Angiography:** The widespread use of multidetector row CT scanners has improved the speed, volume coverage and slice thickness of images so that a single contrast bolus can be imaged as it passes through the arterial system. The advantage is the depiction of the entire vessel with the ability to appreciate thrombus and calcification. Thin slices of 0.625 mm allows for three dimensional reconstructions and multiplanar reformatting.
3. **Magnetic Resonance Angiography:** Contrast –enhanced MRA report a high sensitivity and specificity for demonstrating the degree of stenosis and lesion length, and even superiority in identifying distal target vessels.
4. **Carbon Dioxide Angiography:** Angiography using Carbon dioxide as a contrast medium can be helpful in patients with severe chronic renal insufficiency. Carbon dioxide temporarily displaces the blood in the artery being imaged. Carbon dioxide rapidly dissolves, but 3 to 5 minutes must be allowed to pass between injections.

CT-ANGIOGRAM OF LOWER LIMBS

MR ANGIOGRAPHY

5. **Intravascular Ultrasound:** This provides a transverse, 360 degree image of the lumen of the vessel to be imaged throughout its length and provides qualitative data about the wall anatomy. It has been used in peripheral interventions for opening chronic total occlusions.

MANAGEMENT OF PVD

CONSERVATIVE METHODS:

1. Stoppage of smoking often goes a long way to stop the progression of the disease.
2. Diet should be so planned as to reduce weight in case of obese individuals. The diets containing cholesterol or beta lipo-proteins should be avoided.
3. Raised beta lipo-protein is probably the commonest abnormality found in those suffering from atherosclerosis. Cholestyramine is particularly useful which lowers cholesterol level.

4. In cases of intermittent claudication if the patients are properly explained that walking is not doing any harm, the walking distance often improves. Improvement is seen probably due to establishment of adequate collateral circulation in many patients within a few months.
5. Exercise, if performed within limits , often reduces pain of intermittent claudication and may help in spontaneous cure.
6. Diabetes and Hypertension if present should be treated accordingly. Both these diseases play a considerable role in the development of atherosclerosis.
7. Care of the feet- the part should not be exposed to excessive cold or heat. Wearing shoes with high heel diminish action of calf muscles thus reducing demand of blood supply, so pain is relieved to a greater extent. Pressure to the affected foot particularly over the heel and malleoli should be avoided. Nail cutting should be done

cautiously and at the same time any minor trauma should be avoided.

8. Buerger's position- In this position the head end of the bed is raised and at the same time the foot end of the bed is gradually lowered about 6 inches daily. This increases blood supply to the lower extremity and may improve the condition.
9. Buerger's exercise- In this exercise the affected lower limb is elevated for 2 minutes and then lowered below the bed side for another 2 minutes. This process is repeated several times in one sitting. In a day at least 3 sittings should be performed. This also improves circulation to the affected lower limb.
10. Various analgesics may be used, of which aspirin in dispersible form is probably the best due to its anti-adhesive effect on the platelets.
11. Beta blockers may reduce claudication distance. Aspirin in the dose of 75 mg to 300 mg daily is quite a good

and easily available drug. If there is raised blood lipids, drug treatment should be used to reduce these to normal level. Praxilene (Naftidrofuryl oxalate) may increase claudicating distance by allowing a greater oxygen debt to be incurred. Trental (oxpentifylline) reduces blood viscosity and thus may be of some benefit. Intra-arterial administration of vasodilator drugs (papaverine, lignocaine and duvadilan) at the selective site by injection has been successful as a temporary method.Paravertebral injection of local anaesthetics by the side of the L2, 3 and 4 vertebra near the sympathetic chain has also improved circulation of the lower limb. It is also known as chemical sympathectomy.

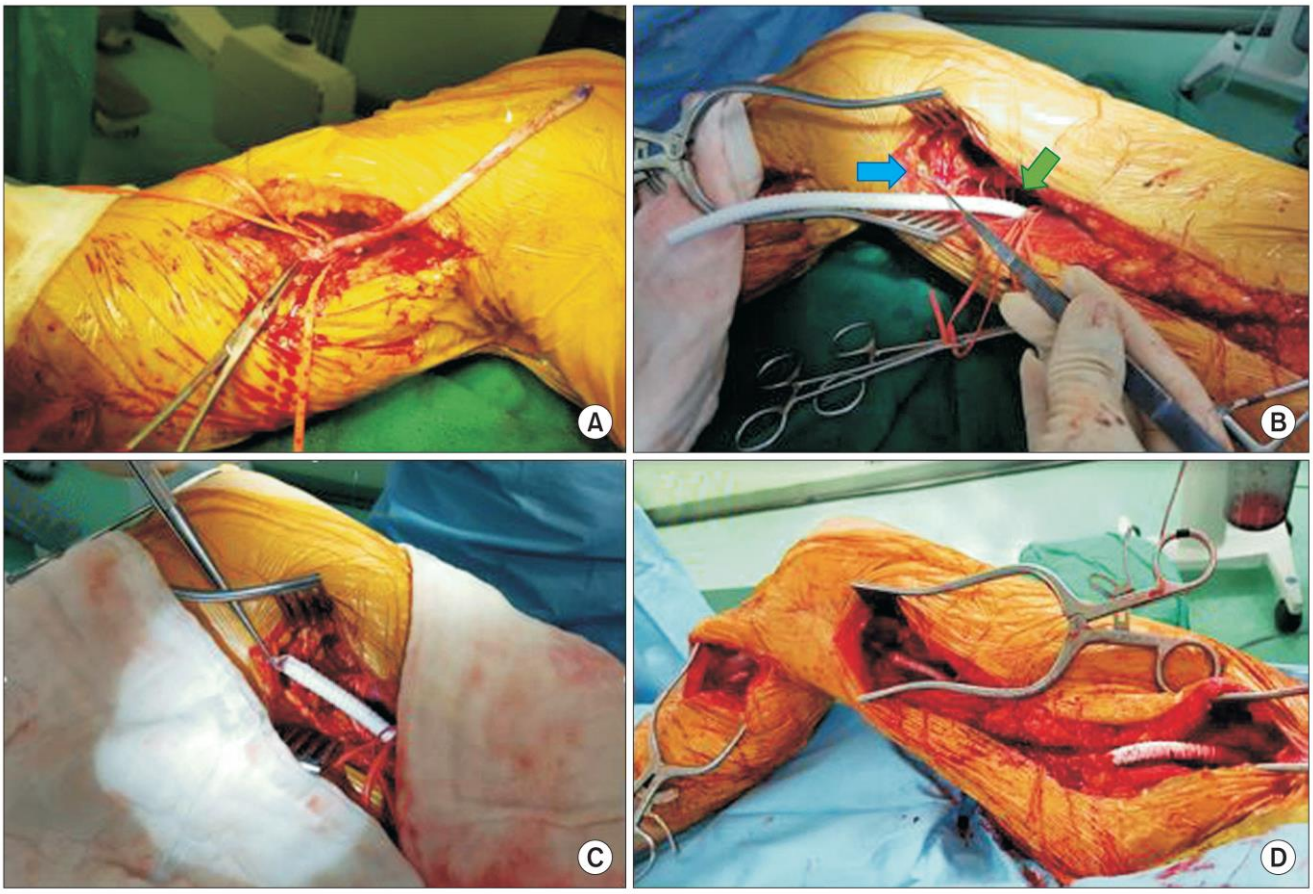
SURGICAL MANAGEMENT:

1. **Thromboendarterectomy:** This operation is performed when the atheromatous lesion is short and localised and has affected a big artery like aorto-iliac occlusion or less

commonly in femoro-popliteal occlusion. Though it may require more extensive operation, yet it has the advantages of preserving the patient's own arteries and avoiding the use of a prosthesis with its attendant risks. This operation is also called 'disobliteration' or 'reboring'.

In this operation, the whole length of the obliterated segment is explored. When the affected segment is small, a longitudinal incision is made over the diseased segment and is made deep till the atheromatous plaque is reached. The diseased intima , the atheromatous plaque and the thrombus are removed through a plane of cleavage through the middle of the tunica media. The arteriotomy is then closed with continuous 5/0 polypropylene (prolene) suture with or without a vein patch graft. This patch graft is to avoid constriction following longitudinal suturing. This is called ''Open endarterectomy''.

ARTERIAL BY-PASS GRAFTING



In case of longer diseased segment, arteriotomies are made only at the upper and lower ends of the diseased segment. An endarterectomy loop is inserted through the lower arteriotomy and is pushed upwards dissecting and separating the atheromatous plaque as far as the upper arteriotomy. The atheromatous plaque is removed from the upper arteriotomy. The arterial openings are closed after ensuring good flow. General anticoagulant therapy is instituted. This is called ''Closed endarterectomy''.

2. **Arterial by-pass grafting:** In this technique the diseased segment is bypassed with a synthetic graft (knitted Dacron or Teflon) or with reversed autogenous long saphenous vein graft. Prosthesis come in two types- woven and knitted. Woven grafts leak less when first exposed to blood during surgery. Modern knitted

prosthesis leak even less as they are sealed with gelatin or collagen by the manufacturer. In aorto-iliac occlusion mostly synthetic grafts are applied, whereas in femoro-popliteal occlusion, autogenous vein graft is mainly used. A polytetrafluoroethylene(PTFE) graft may be used instead of autogenous vein graft. This often gives prolonged patency. In case of aorto-iliac occlusion suture material used is monofilament polypropylene-2/0 or 3/0. In femoro-popliteal occlusion at the groin, 4/0 or 5/0 polypropylene is used; whereas in case of further down limb occlusion 7/0 suture material is used.

3. **Balloon transluminal angioplasty:** In this technique a balloon angioplasty catheter is passed through the arterial stenosis. The balloon is inflated with dilute contrast medium to a pressure of 5 to 10 atmospheres for a period of 15 to 30 seconds even up to 1 minute, after which it is deflated. This is repeated for a few times before withdrawal of the catheter. This balloon is

introduced into the arterial tree using the Seldinger technique. Expansion of the balloon produces fissures in atheromatous plaques and also ruptures muscle fibres of the tunica media thus widening the lumen and ensuring blood supply to the distal limb. This technique is mainly used in case of arterial occlusions of the iliac artery, superficial femoral or renal artery. Gradually the endothelial lining develops along the fissures in the atheromatous plaque within a few months.

4. **Transluminal angioplasty and stenting**: This is particularly effective to inflate a narrowed short occlusion. The balloon catheter is inserted over a guide wire. The balloon is positioned within the stenosis or occlusion which is confirmed by angiography. The problem is that often the vessel fails to stay adequately dilated after such treatment and in these cases metal stent may be used. In this technique, the balloon catheter is introduced through the expanding stent and

then the balloon is inflated. The stent becomes rigid after being expanded and keeps the vessel widely patent. The catheter balloon is now deflated and the catheter is removed. There is also a type of self-expanding stent, which is held compressed by a sheath of plastic before application. The stent is positioned at the site of stenosis and the plastic sheath is withdrawn. The stent self-expands and holds the vessel lumen wide open.This procedure is not as good as reconstructive surgery. The advantage of this procedure is that it can be repeated if stenosis recurs.

5. **Other operative salvage procedures**: These operations should not be performed for intermittent claudication as gangrene or loss of limb may result if these operations fail.

   (a) Femoro-femoral cross over graft- In this technique without extensive exploration and minimizing the operation time, an iliac artery occlusion may be

relieved if the other iliac artery is patent with a strong femoral pulse. 8mm Dacron graft is placed in a tunnel in the subcutaneous tissue in the lower abdomen just above the pubis from one groin to the other to connect the common femoral arteries on each side. So that blood from the patent femoral artery is carried through the graft to the other femoral artery of the ischemic side.

(b) Hitch-hike femoro-popliteal-tibial graft- This technique is adopted in cases of difficult femoro-popliteal occlusion. A Dacron graft connects the common femoral artery to a thromboendarterectomised upper popliteal artery. A vein graft is again connected from this rebored upper popliteal artery to the arteries below the knee.

(c) Axillo-femoral graft- This technique is used for the pre-gangrenous limb in a poor risk patient with bilateral iliac obstructions. 8 mm Dacron graft is

placed in a tunnel in the subcutaneous tissue extending from the axilla to the groin. In its upper part it is anastomosed with the axillary artery in an end-to-side fashion. In its lower end it is anastomosed to the femoral artery of the involved limb in the same fashion. By this, blood flows sufficiently from the axillary artery to revascularise the lower limb.

DIABETIC FOOT

PVD is common among patients with diabetes. Intermittent claudication is twice as common among diabetic patients than among non-diabetic patients. An increase in HbA1c by 1% can result in more than a 25 % risk of PVD. Major amputation rates are five to ten times higher in diabetics than non-diabetics. Because of these causal relations, the American Diabetes Association

recommends ABI screening every 5 years in patients with diabetes.

The care of diabetic patients should start with preventive measures and it is important to avoid infections in patients with insensate feet because of neuropathy. These patients need to wear properly fitted shoes at all times for protection. Orthotic inserts should be used to distribute weight evenly to avoid pressure on the metatarsal heads of the foot. In this population, infections can progress rapidly, with significant tissue damage from a combination of delayed presentation and compromised immune function.

On presentation, a careful physical examination is important to plan for appropriate treatment. The overlying cellulitis is assessed and any possible underlying abscess is examined by palpation for crepitus or detection of drainage of purulent

fluid. Cellulitis should not be confused with dependent rubor caused by severe ischemia in patients with PVD. The presence of an abscess requires immediate drainage prior to revascularization. The presence or absence of lower extremity pulses in the common femoral, popliteal and pedal arteries is examined. The pulses may be difficult to palpate because of swelling from foot infection; non-invasive arterial ultrasound can be useful in assessing the extent of arterial disease.

Insulin-dependent diabetic patients may have calcified walls of the medium and small arteries that can falsely elevate the segmental pressures of the leg. In this situation, digital pressures of the toes can be accurately measured and a pressure higher than 30 mm Hg is predictive of healing after local amputation and debridement.

Plain x-rays with multiple views of the foot can assist in assessing the extent of foot infection. Gas in soft tissue signifies deep tissue infection and the need for urgent surgical debridement. Advanced osteomyelitis can be detected; however, plain films may not show early bone infection. Magnetic resonance imaging (MRI) of the foot is a sensitive imaging modality for detecting soft tissue infection and early osteomyelitis.

In infections with only cellulitis and no underlying soft tissue involvement, patients are treated with cellulitis does not resolve in several days, there may not be adequate IV antibiotic therapy. If the antibiotic coverage and the presence of deep tissue infection is considered. The choice of the antibiotics used and the foot need to be re-evaluated; reimaging the foot may be necessary.

The cause of persistent cellulitis and non-healing infection is usually underlying deep infection or osteomyelitis. Other patients may present with gangrene, open joint or exposed bone, or abscess. In these patients, surgical debridement is required in addition to antibiotic therapy. Small open wounds can be treated with simple debridement, but often there is deep tissue involvement that is not visible on the surface. To remove all non-viable tissue and wide drainage, amputation may be required. If there is extensive infection of the foot with gas, calf pain or systemic sepsis, the patient may require amputation as an initial therapy. After surgical debridement, patients are treated with aggressive wound care using dressing changes and continued broad-spectrum antibiotic therapy until intra-operative culture sensitivities are finalized and allow for the use of targeted anti-microbials. Wounds are evaluated

closely for persistent infection that may require additional surgical intervention. In patients with adequate arterial circulation, the wound can be closed secondarily after resolution of the infection.

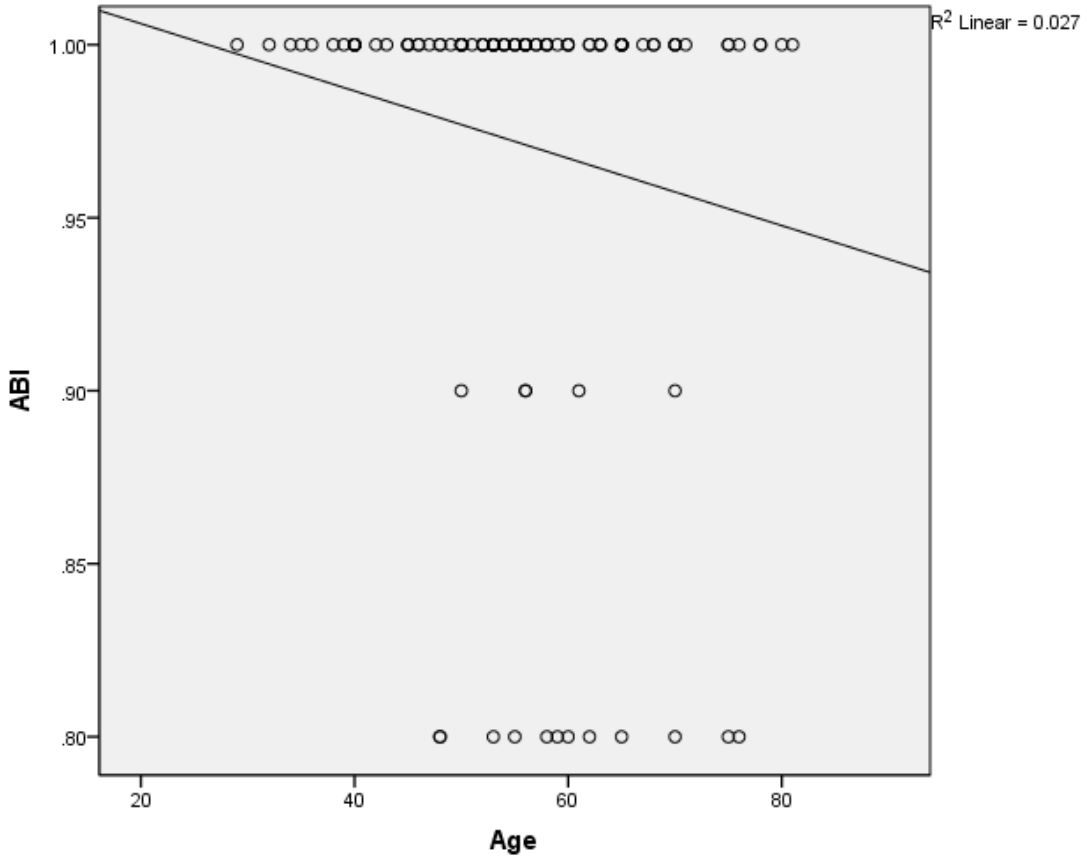
All patients with evidence of concomitant arterial occlusive disease are considered for lower extremity revascularization with open bypass surgery or endovascular stenting or angioplasty to optimize wound healing and limb salvage.

MATERIALS AND METHODS

- This is a cross-sectional study conducted in Government Kilpauk Medical College, Kilpauk , Chennai-600 010 during the period of December 2016 to September 2017.
- The study included 100 diabetic patients with foot ulcers who attended the Surgical Outpatient Department
- The patients were duly informed about the nature of the study and after getting consent from the patients in the language (Tamil) which they can read/understand the Demographic, Clinical and Laboratory details are collected.
- Ankle-Brachial Index (ABI) was measured for each individual patient using the portable hand held Doppler. ABI < 0.9 is taken as the cut-off to identify the patient as having Peripheral Vascular Disease (PVD).

OBSERVATION &RESULTS

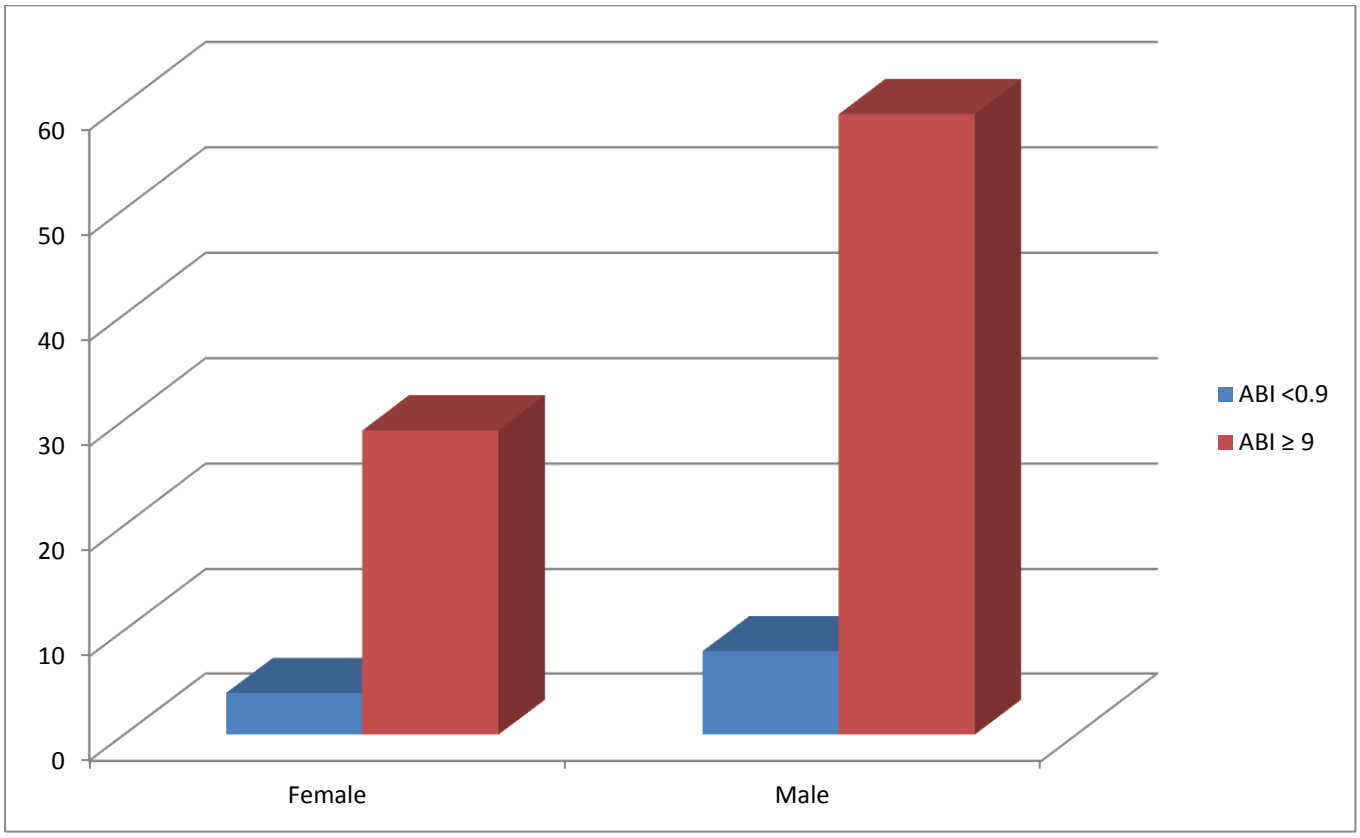
Data is entered in Microsoft Excel and analysed by using SPSS version 20.0. Descriptive Statistics such as Frequency, proportion, mean and standard error are used to describe the data. Inferential statistics such as Chisqaure test, Pearson correlation were used to analyse the data.



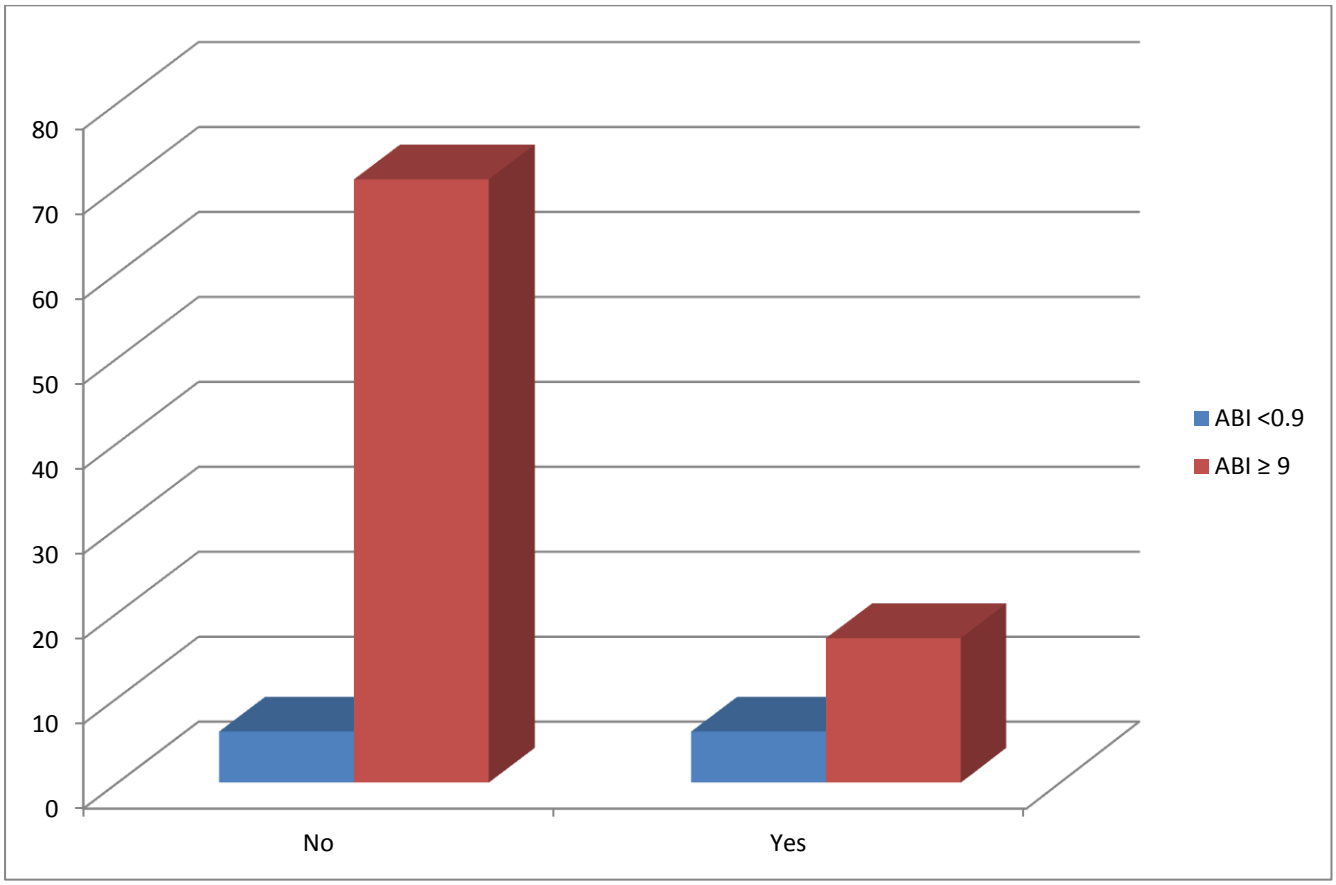
Pearson correlation coefficient -0.165

P value – 0.101

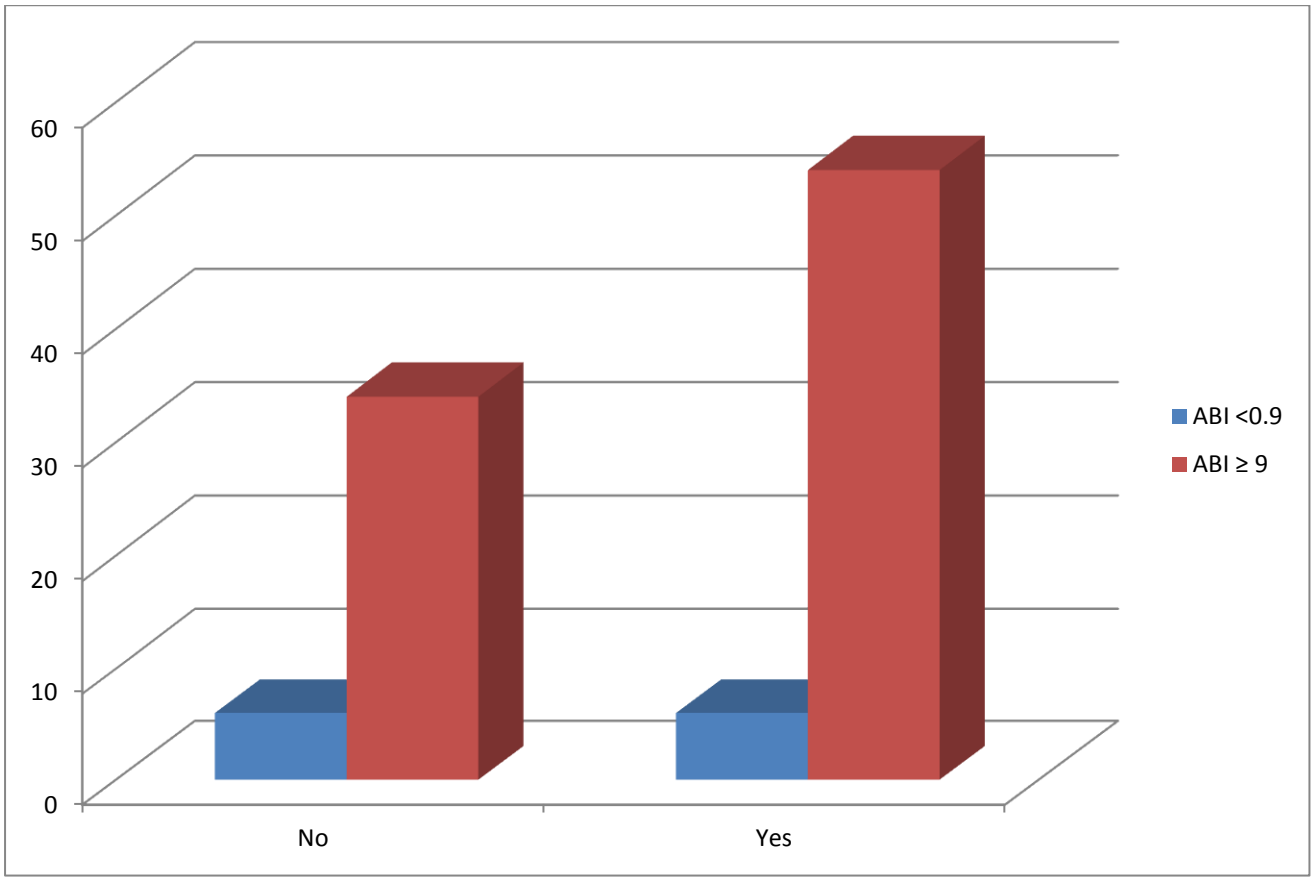
Sex	ABI <0.9	ABI ≥ 9	Fisher exact probability	P value
Female	4	29	0.001	1.00
Male	8	59		

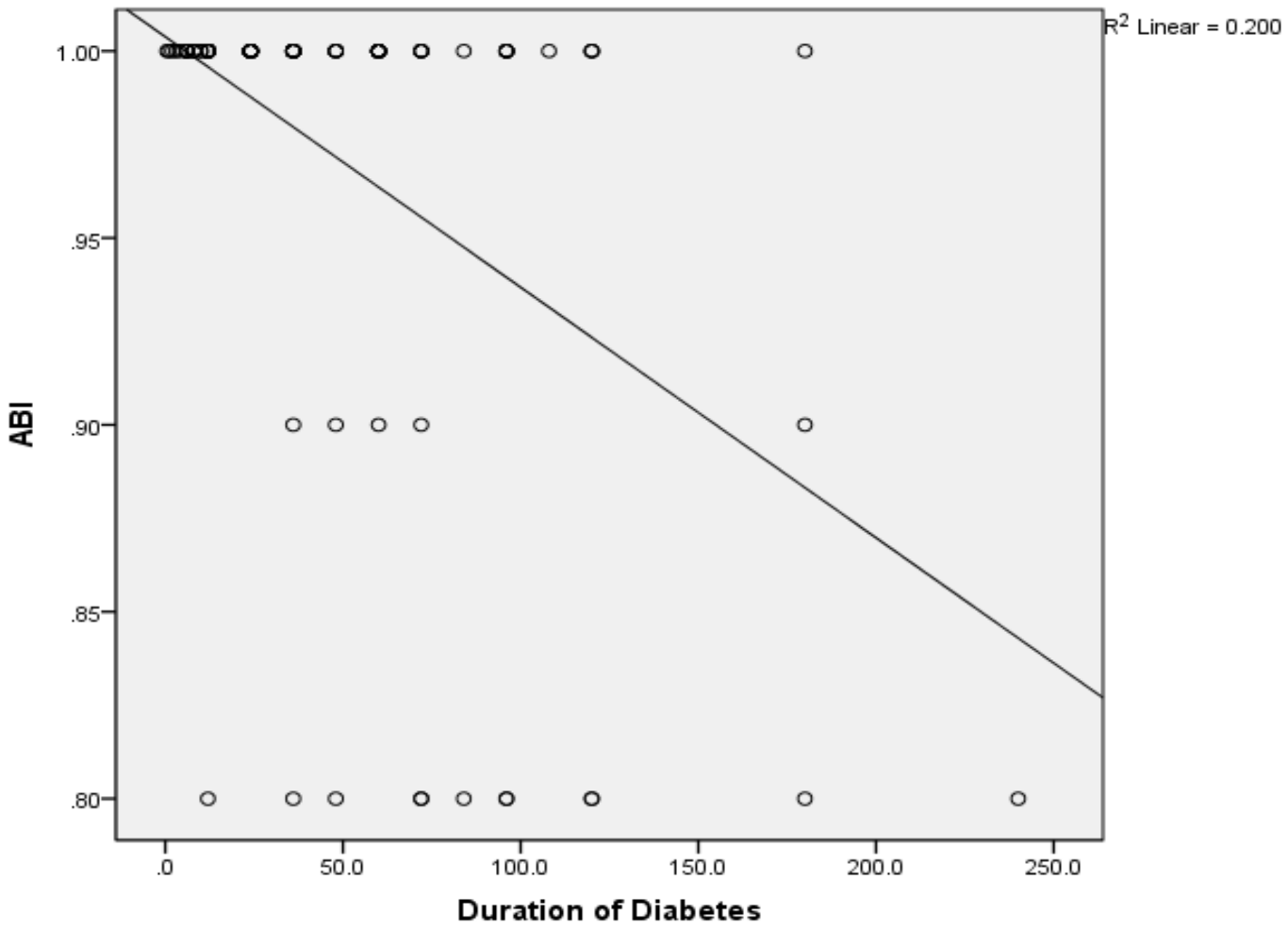


On Insulin	ABI <0.9	ABI ≥ 9	Fisher exact probability	P value
No	6	71	5.613	0.028
Yes	6	17		



On OHA	ABI <0.9	ABI ≥ 9	Fisher exact probability	P value
No	6	34	0.568	0.535
Yes	6	54		

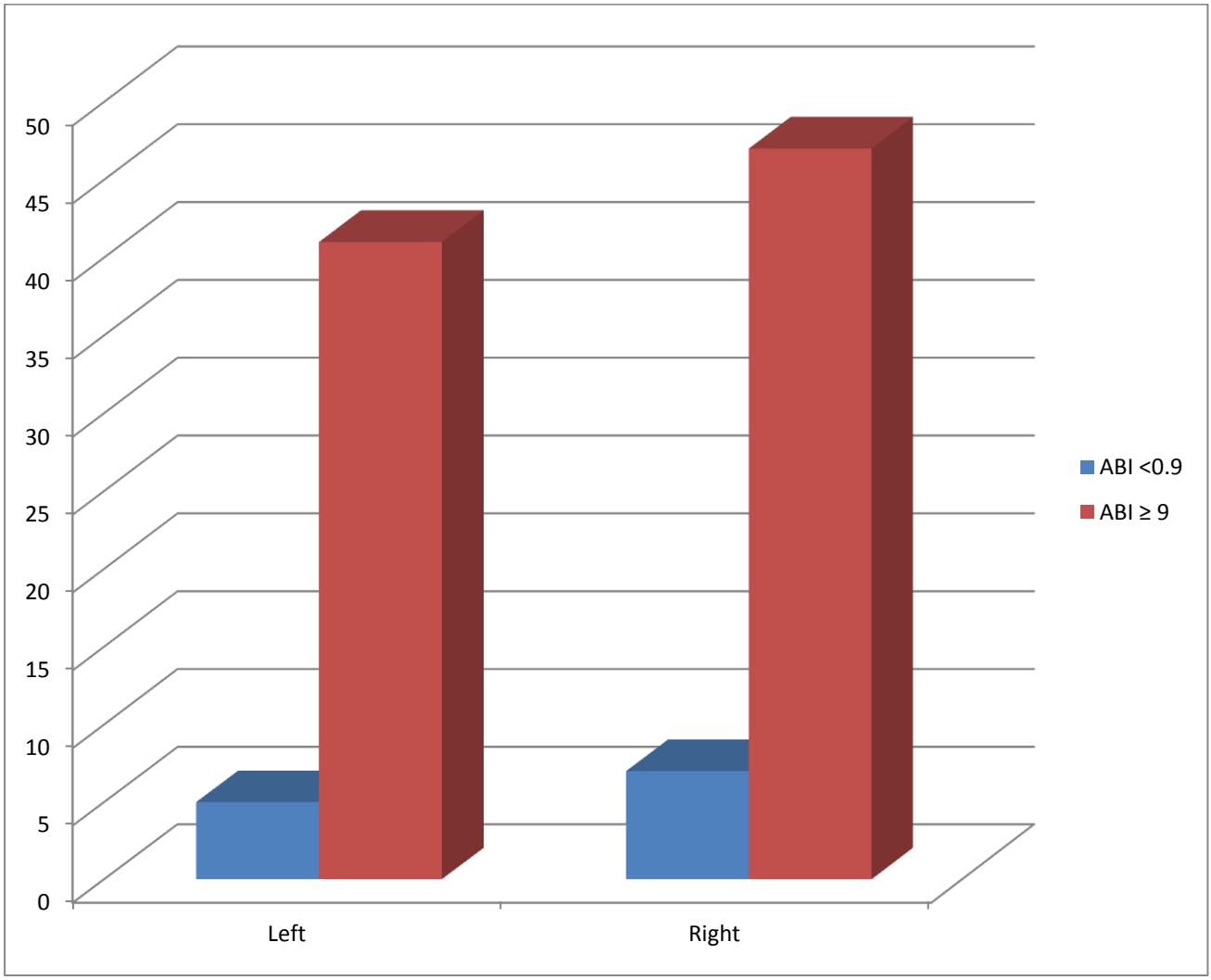




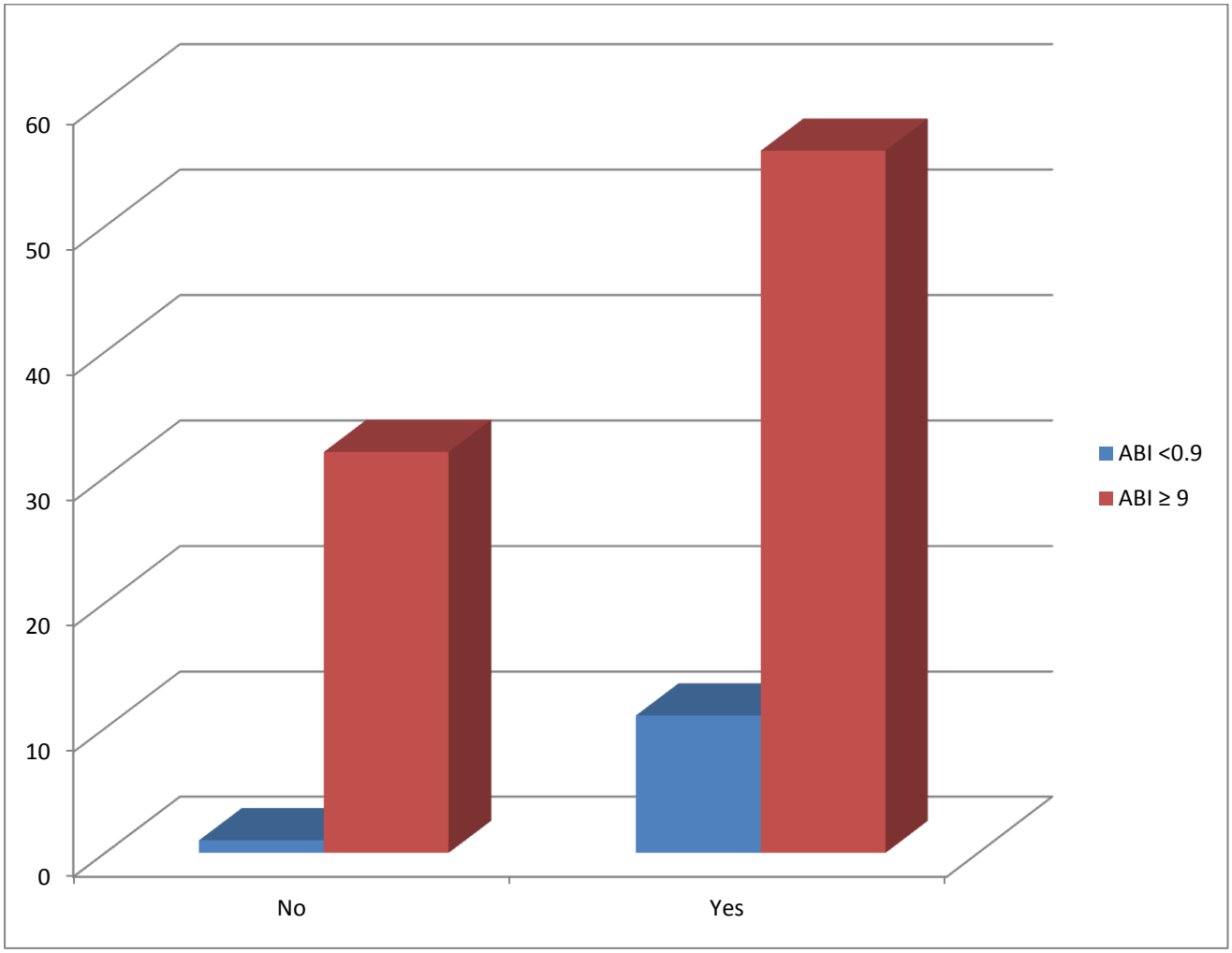
Pearson correlation -0.447

P value – 0.0001

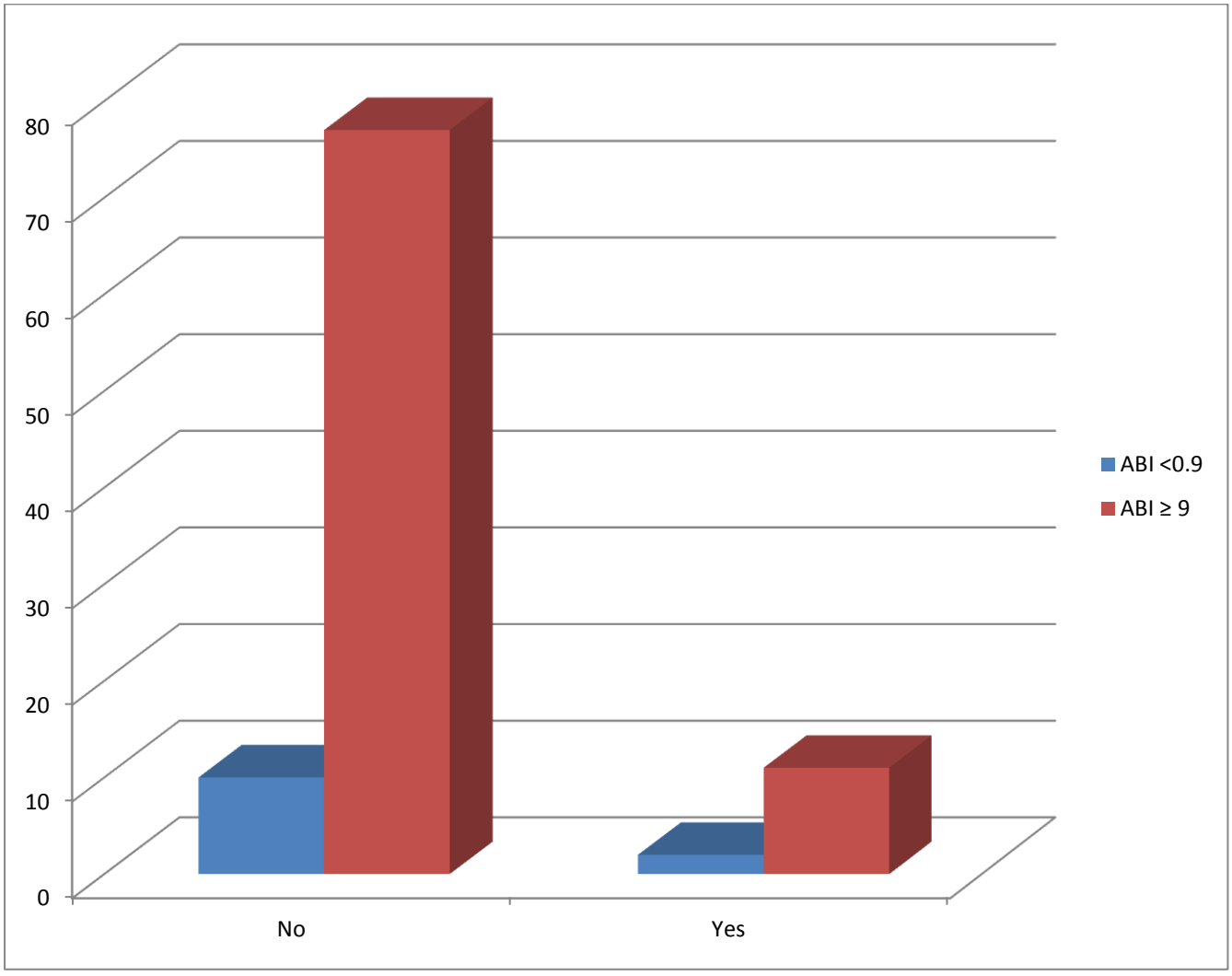
Foot involved	ABI <0.9	ABI ≥ 9	Chisquare	P value
Left	5	41	0.103	0.748
Right	7	47		



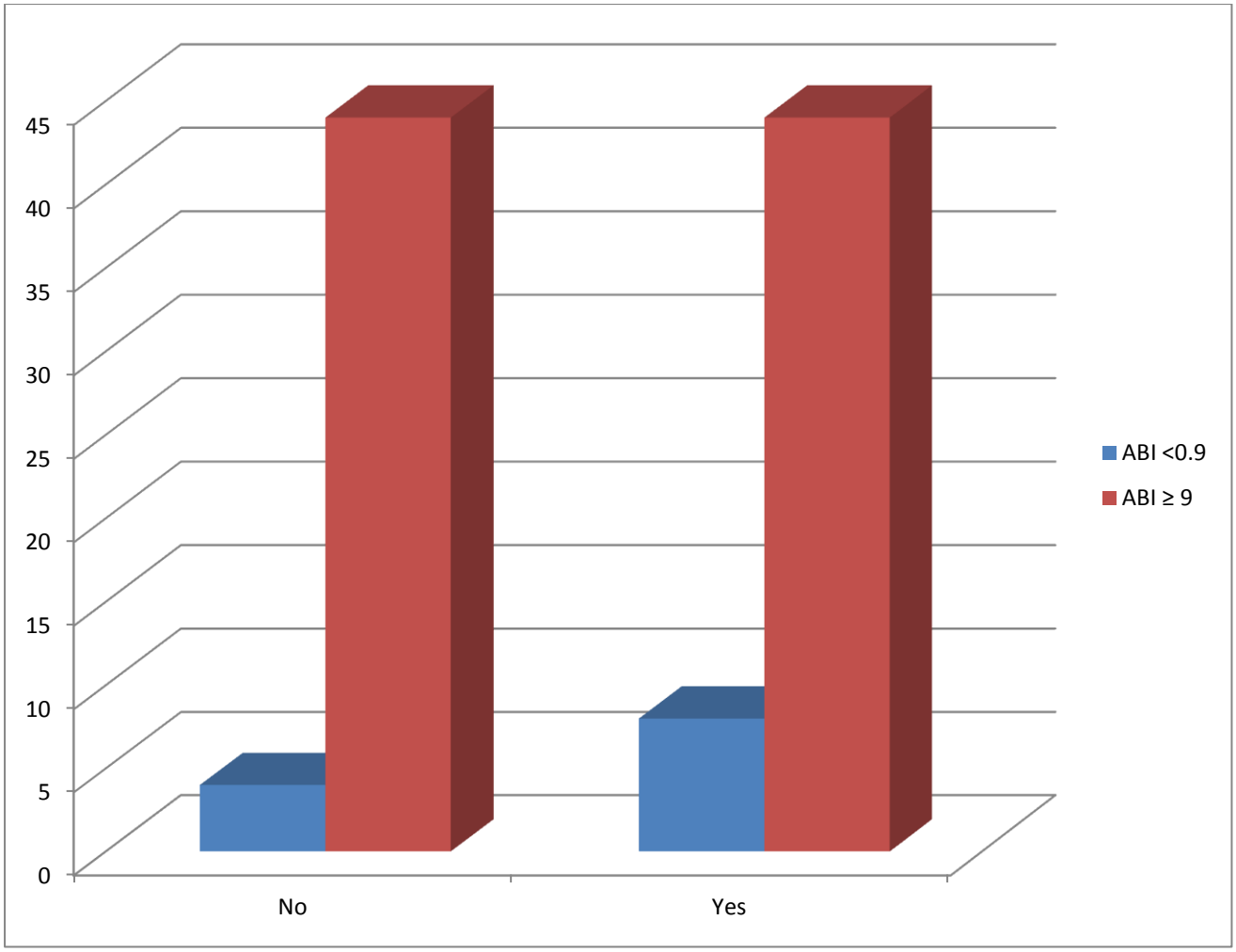
Trauma	ABI <0.9	ABI ≥ 9	Fisher exact probability	P value
No	1	32	3.753	0.097
Yes	11	56		



Cellulitis	ABI <0.9	ABI ≥ 9	Fisher exact probability	P value
No	10	77	0.162	0.653
Yes	2	11		

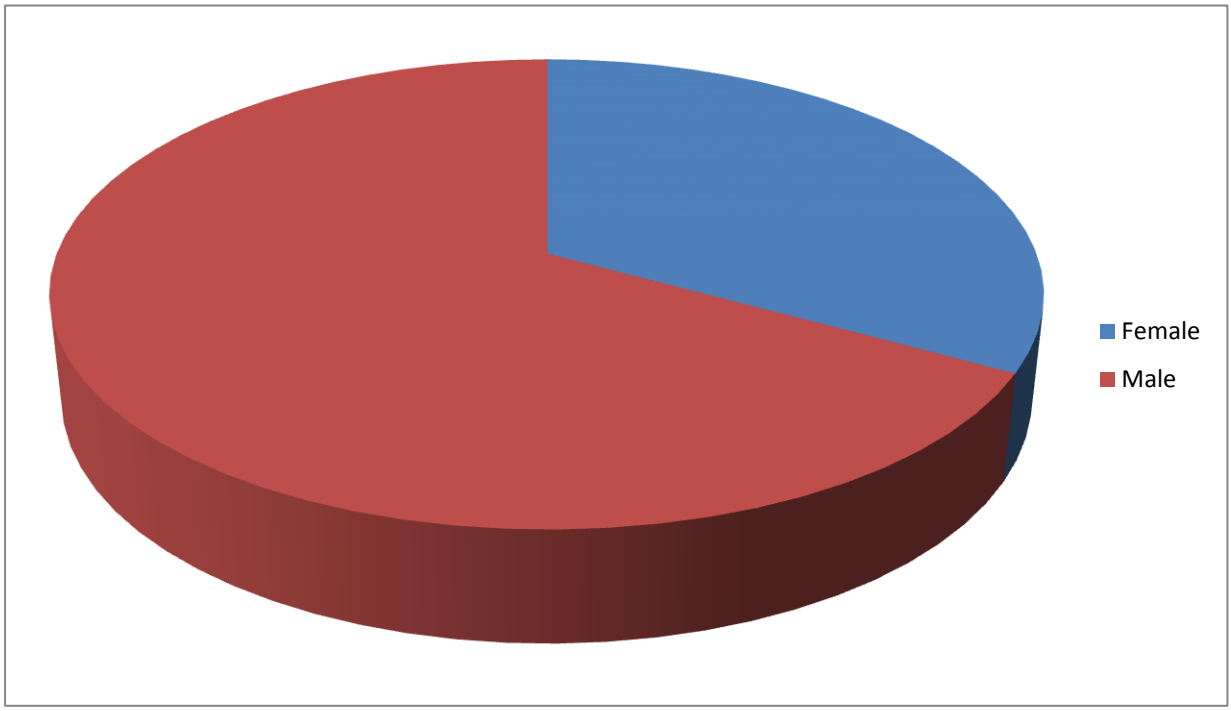


Discharge	ABI <0.9	ABI ≥ 9	Chisquare	P value
No	4	44	1.175	0.278
Yes	8	44		



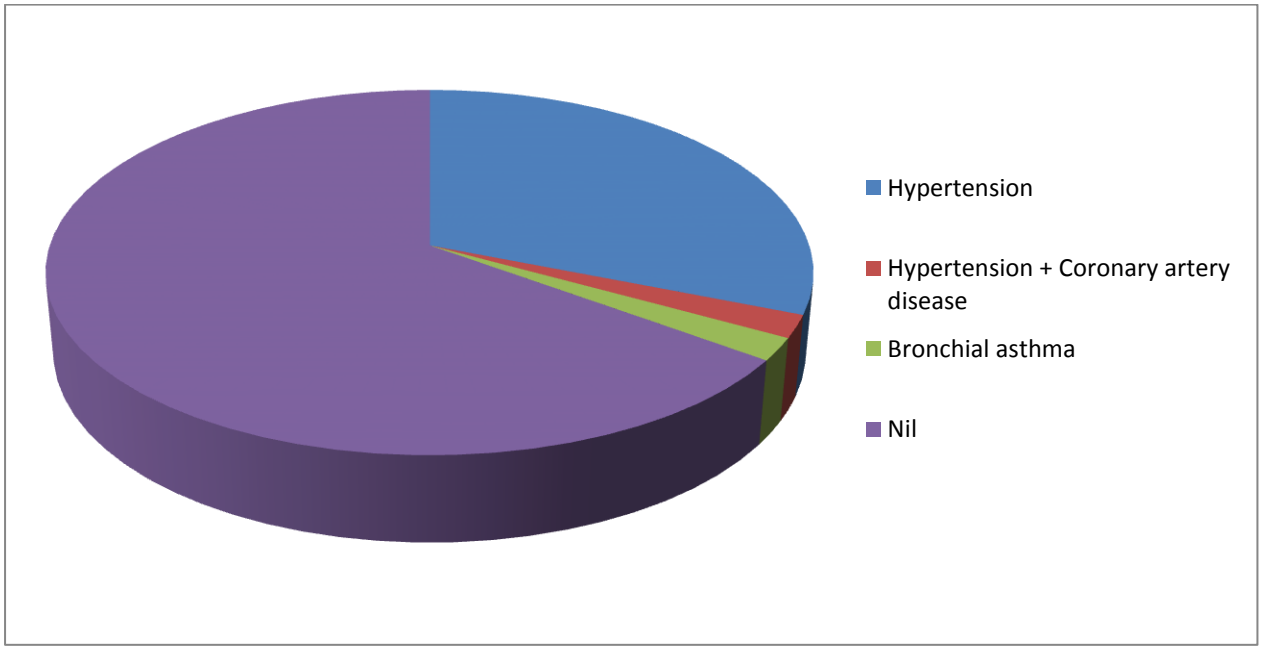
DISCUSSION

In this study of 100 diabetic patients with foot ulcers who attended the Surgical Outpatient Department, 67 were male and 33 were female.



About 23 patients were on treatment with Insulin and 60 patients were on treatment with Oral Hypoglycemic Agents. 17 patients were found to had no treatment for diabetes.

About 65 patients had no co-morbid conditions other than diabetes. 31 patients had associated Hypertension. 2 patients had associated Bronchial Asthma. 2 patients had associated Hypertension and Coronary Artery Disease.



Right foot was involved in about 54 patients while left foot was involved in 46 patients.

History of trauma was present in 67 patients.

13 patients presented with cellulitis and 52 patients had active discharge from the foot ulcers.

The youngest in the study population was 29 years and the eldest was 81 years. The duration of diabetes ranges from 2 weeks to 20 years. ABI was found to be 1 in majority of patients (83), 0.9 in 5 patients and 0.8 in 12 patients. Taking into consideration of ABI <0.9 as the cut-off value 12 patients were found to have Peripheral Vascular Disease (PVD).

Among the patients identified to have Peripheral Vascular Disease (12) majority of patients (9) had duration of diabetes ≥ 6 years and about half of the patients (6) were on treatment with Insulin for uncontrolled diabetes; rest of the patients (6) were on treatment with Oral Hypoglycemic Agents and glycemic status during study was found to be under control in 8 patients (5 on insulin therapy and 3 on OHAs respectively).

CONCLUSION

- The prevalence of Peripheral Vascular Disease (PVD) in this cross sectional study conducted in 100 diabetic patients with foot ulcers who attended the Surgical Outpatient Department at Kilpauk Medical College Hospital, Kilpauk, Chennai in the period of December 2016 to September 2017 was found to be 12 %
- The increased duration of diabetes with uncontrolled glycemic status was found to have a significant role in the development of Peripheral Vascular Disease and its complications in diabetic population.

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PROFORMA

NAME:

AGE/SEX:

Duration of DIabetes	
Anti-Diabetic medications	
Fasting Blood sugar	
Post prandial Blood sugar	
Urine sugar	
Urine Acetone	

ULCER STATUS:

Size and extent	
Discharge	
Associated cellulitis	
Associated skin lesions	
History of trauma	
History of similar ulcers before, If yes, details of treatment	

PERIPHERAL VASCULAR STATUS:

	Rt.	**Lt.**
Femoral Artery		
Popliteal artery		
Anterior Tibial Artery		
Dorsalis Pedis Artery		
Posterior Tibial Artery		

ANKLE BRACHIAL PRESSURE MEASUREMENT:

	Rt.	**Lt.**
Brachial systolic pressure		
Ankle systolic pressure		

Anklle-Brachial Pressure Index (ABI):

சுயஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

காலில் ஆறாப்புண் ஏற்பட்டுள்ள சர்க்கரை நோயாளிகளின் காலின் இரத்தக் குழாய் அடைப்பு நோய் குறித்த ஒரு ஆய்வு

A Study on the Prevalence of Peripheral Vascular Disease in Diabetic Foot Ulcer Patients

ஆய்வு செய்யப்படும் துறை : பொது அறுவை சிகிச்சை துறை

மருத்துவமனை : அரசு கீழ்பாக்கம் அரசு மருத்துவ கல்லூரி மருத்துவமனை

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் வயது :

பங்கு பெறுபவரின் மருத்துவமனை எண்.:

பங்கு பெறுபவர் இதனை (✓) குறிக்கவும்:

1. Ankle-Brachial pressure Index (ABI) என்னும் பரிசோதனை மூலம் சர்க்கரை நோயாளிகளின் காலின் ரத்த குழாய் அடைப்பு நோய் குறித்து ஆரம்ப நிலையிலேயே கண்டறிய முடியும் என்பது பற்றி எனக்கு தெளிவாக விளக்கப்பட்டது. மேலும் என்னுடைய சந்தேகங்களை கேட்கவும் அதற்கான விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது என அறிந்து கொண்டேன். ( )
2. நான் இந்த ஆய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ நான் இந்த ஆய்வில் இருந்து விலக ஆசைப்பட்டால் எந்த பிரச்சனையும் இன்றி விலகலாம் என்றும் அறிந்து கொண்டேன். ( )
3. இந்த ஆய்வு சம்மந்தமாகவோ இவை சார்ந்த மேலும் ஆய்வு மேற்க்கொள்ளும் பொழுதோ இந்த ஆய்வில் பங்கு பெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்தேன். ( )
4. இந்த ஆய்வில் பங்கு கொள்ள நான் சுயநினைவோடும், முழு சம்மதத்தோடும் ஒப்புதல் அளிக்கிறேன். ( )

பங்கு பெறுபவரின் பெயர் :

ஆய்வாளரின் பெயர்:

பங்கு பெறுபவரின் கையொப்பம் :

ஆய்வாளரின் கையொப்பம்:

Sl.No.	Name	Age/sex	O.P. No.	Duration of Diabetes	Co-morbidities	Foot	Trauma	Cellulitis	Discharge	ABI
1.	Ashadevi	46/F	43901	2 years	-	R	+	-	-	1
2.	Varalakshmi	38/F	43912	4 months	-	L	+	+	+	1
3.	Dominic	52/M	43954	3 years	HT,CAD	R	+	-	-	1
4.	Lalitha	75/F	45832	8 years	HT	L	-	-	+	1
5.	Kannadhasan	55/M	46382	10 years	-	R	+	+	+	1
6.	Padmavathy	68/F	44213	1 month	-	L	+	-	-	1
7.	Raman	70/M	48234	20 years	-	R	+	-	+	0.8
8.	Damodharan	52/M	42115	5 years	HT	R	+	-	-	1
9.	Selvi	56/F	42312	10 months	-	L	+	-	-	1
10.	Sridhar	48/M	45623	3 years	-	L	+	+	+	0.8
11.	Hemamalini	32/F	32453	1 year	-	L	-	-	-	1
12.	Saraswathy	65/F	34213	4 years	HT	R	+	-	-	1
13.	Rajalingham	53/M	42312	1 year	-	R	+	-	-	1
14.	Bhavani	65/F	42352	8 years	HT	L	-	-	-	0.8
15.	Rajamani	40/F	41112	3 years	BA	R	-	+	+	1
16.	Selvam	40/M	42333	2 years	-	L	+	-	+	1
17.	Nagammal	65/F	45213	6 months	HT	R	+	-	+	1
18.	Radha	60/F	42231	7 years	-	L	+	-	-	1
19.	Angel	40/F	32143	3 months	-	L	-	-	-	1
20.	Shanthi	53/F	35214	5 years	HT	L	-	-	-	1
21.	Prabhu	29/M	42136	1 year	-	R	+	-	-	1
22.	Jayaraman	75/M	21334	15 years	-	L	+	-	-	0.8
23.	Ramesh	40/M	32453	1 year	-	L	-	-	-	1
24.	Ramasamy	80/M	42351	6 months	-	R	+	-	-	1
25.	Jayabalan	63/M	32453	5 years	HT	L	-	-	+	1

Sl.No.	Name	Age/Sex	O.P.No	Duration of Diabetes	Co-morbidities	Foot	Trauma	Cellulitis	Discharge	ABI
26.	Velmurugan	43/M	21345	2 years	-	R	+	-	-	1
27.	Vasantha	55/F	42314	2 years	HT.CAD	L	+	-	+	1
28.	Menaga	59/F	42222	6 years	-	R	+	+	+	0.8
29.	Kumar	56/F	41623	4 years	-	L	-	-	+	0.9
30.	Nagaraj	62/M	22674	5 years	HT	L	-	-	-	1
31.	Selvakannan	34/M	22567	1 year	-	R	+	-	+	1
32.	Jayalakshmi	45/F	32145	1year	-	R	+	-	-	1
33.	Alamelu	61/F	42178	6 years	-	R	+	+	+	0.9
34.	Gowsdheen	65/M	32156	9 years	HT	L	+	-	-	1
35.	Vajravel	54/M	43278	6 years	HT	L	+	-	+	1
36.	Devan	76/M	32456	10 years	-	R	+	+	+	1
37.	Kasthuri	75/F	23831	6 months	HT	L	-	-	+	1
38.	Shakila	40/F	45385	5 years	-	R	-	+	+	1
39.	Sekar	56/M	32454	8 years	HT	L	+	-	+	1
40.	Anandh kumar	53/M	32144	8 years	HT	R	+	-	-	1
41.	Ganesan	50/M	23342	2 years	-	R	-	-	-	1
42.	Baskar	36/M	42333	8 months	-	R	+	-	-	1
43.	Ponnamal	58/F	41115	2 weeks	-	R	-	-	-	1
44.	Lakshmi	52/F	22376	2 months	-	L	+	-	+	1
45.	Saravannan	35/M	34821	1 year	-	R	+	-	-	1
46.	Durairaj	50/M	52315	3 years	HT	R	-	-	+	1
47.	Ravi kumar	51/M	42665	8 years	-	L	-	-	+	1
48.	Subramaniyam	62/M	42772	3 years	HT	R	+	-	+	1
49.	Chakkarapani	54/M	22443	4 years	-	R	+	-	-	1
50.	Fathima Mary	50 /M	54432	2 years	HT	R	-	-	+	1

Sl.No.	Name	Age/Sex	O.P.No	Duration of Diabetes	Co-morbidities	Foot	Trauma	Cellulitis	Discharge	ABI
51.	Venilla	39/F	52312	1 year	-	R	+	+	+	1
52.	Murali	46/M	53241	5 years	-	R	+	-	+	1
53.	Kandhasamy	76/M	55323	7 years	-	R	+	-	+	0.8
54.	Rajan Gupta	62/M	56232	8 years	HT	L	+	-	+	0.8
55.	Kannan	78/M	52133	2 years	-	L	-	-	-	1
56.	Valliyamal	54/F	52332	2 years	-	L	+	-	-	1
57.	Rahman khan	57/M	42132	6 years	HT	L	-	-	-	1
58.	Veeraraghavan	56/M	32216	6 years	HT	L	+	-	-	1
59.	Nagomi	70/F	42667	15 years	HT	R	-	+	+	0.9
60.	Parimala	42/F	52231	3 years	-	R	+	-	-	1
61.	Velayudham	78/M	32642	10 months	-	R	+	-	+	1
62.	Jayaraman	71/M	23345	1 year	HT	R	+	-	+	1
63.	Kumar	48/M	34235	4 years	-	R	+	-	-	0.8
64.	Masilamani	65/M	44233	3 years	HT	R	+	-	+	1
65.	Rama	59/F	52342	8 months		R	+	-	-	1
66.	Kuppan	60/M	52222	1 year	-	R	+	-	+	0.8
67.	Chandrasekar	68/M	33444	5 years	HT	L	-	-	-	1
68.	Nagavalli	53/F	41896	10 years	-	R	+	-	-	0.8
69.	Devagi	55/F	43562	6 years	HT	L	+	-	+	0.8
70.	Krishnan	63/M	52342	5 years	HT	R	+	-	+	1
71.	Kannan	65/M	26443	2 years	-	R	-	-	-	1
72.	Jayashankar	49/M	26591	5 years	-	R	+	-	-	1
73.	Chandrasekar	58/M	26543	10 years	HT	R	+	-	+	0.8
74.	Ashok kumar	56/M	34455	1 year	HT	L	+	-	+	1
75.	Balaji	47/M	45223	6 months	-	L	-	-	-	1

Sl.No.	Name	Age/Sex	O.P.No.	Duration of Diabetes	Co-morbidities	Foot	Trauma	Cellulitis	Discharge	ABI
76.	Ravichandran	48/M	42445	3 years	-	L	-	-	-	1
77.	Rathinavel	63/M	23442	6 years	-	R	+	-	+	1
78.	Nithya kumar	58/M	23546	1 year	-	R	+	-	+	1
79.	Karunakaran	50/M	52341	3 years	-	R	+	-	-	0.9
80.	Abirami	70/F	42332	5 years	BA	R	+	+	+	1
81.	Susidharan	58/M	52334	2 years	-	L	+	-	+	1
82.	Rajeshwaran	50/M	52875	2 years	-	L	+	-	+	1
83.	Venkatraman	65/M	35613	2 years	-	R	+	-	-	1
84.	Elangovan	60/M	45567	8 months	-	L	+	-	-	1
85.	Kali	60/M	52345	3 years	HT	L	+	+	+	1
86.	Dhavamani	53/F	36788	6 months	HT	L	-	-	-	1
88.	Narayannan	67/M	52345	3 years	-	L	-	-	+	1
89.	Muniyammal	81/F	56762	10 years	HT	R	-	-	+	1
90.	Balaji	56/M	43556	5 years	-	L	-	-	-	0.9
91.	Siddhique Ali	70/M	23422	15 years	HT	R	+	-	+	1
92.	Naganathan	57/M	53443	1 year	HT	L	+	-	+	1
93.	Mohammed Ali	53/M	53449	3 years	-	R	+	-	-	1
94.	Rathinam	70/M	45566	5 years	-	R	+	-	+	1
95.	Palani	48/M	29921	4 years	HT	R	-	-	-	1
96.	Saravannan	45/M	34457	2 years	-	R	-	-	+	1
97.	Anandh kumar	53/M	26673	2 years	-	L	-	-	-	1
98.	Malliga	55/M	53432	8 years	HT	L	+	-	+	1
99.	Palani	56/M	53442	4 years	-	L	-	-	-	1
100.	Sampath kumar	50/M	26632	3 years	-	L	+	+	+	1